Research Square

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Bilateral SDH with Sepsis Due to Citobacter Koseri Mimicking Stroke

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Abstract

Central Nervous System (CNS) infections are severe post operative complication of craniotomy, especially in elderly patients that are already immunologically compromised resulting in prolonged management of primary pathology due to active infection and poorer treatment outcomes. The interplay between the primary pathology and the development of a severe infection makes management challenging.. Abrupt onset of unilateral weakness and speech disturbance is important feature of stroke but there are several non vascular conditions which mimic stroke. Severe sepsis is one of these conditions and can lead to cerebral dysregulation, which in turn causes brain hypoperfusion and a stroke-like picture. This is a critical reminder that a comprehensive evaluation, including infection workup, is essential when new neurological deficits appear in post-operative patients. We report one such operated case of bilateral SDH with sepsis due to Citrobacter koseri presenting with stroke like symptoms. In this case, the organism caused severe invasive infection, likely contributing to sepsis, brain hypoperfusion, and potentially exacerbating the patient's neurological status. The recognition of such infections is crucial because their presentation may mimic more common post-operative complications like stroke, leading to potential misdiagnosis and delays in appropriate treatment. The emergence of Citrobacter koseri as a cause of severe invasive infection in neurosurgical patients underscores the need for heightened awareness of both unusual pathogens and atypical presentations. This also highlights the importance of early detection, appropriate antibiotic therapy, and multi-disciplinary management in reducing morbidity and mortality associated with these infections.

1. Introduction

Severe systemic infection can produce stroke like symp- toms especially in patient with vascular risk factor. Citrobac- ter Koseri is a commensal organism found in air, water, soil, occasional colonizer of human and animal gastrointestinal tract. Early diagnosis and treatment is crucial in CNS infec- tions. When infection is masked by stroke like symptoms diagnosis etiological diagnosis become extremely challenging especially in immunocompromised patients, as the ef- fects of the infection combine with those of the underlying disease. Clinical manifestation in such cases is diverse and usually unspecific. We present a case of a 70 year male, background of hypertension, IHD, DM diagnosed with bilat- eral SDH and was operated for same. However during hospi- tal stay, patient developed sudden of left sided weakness and slurring of speech, he was initially diagnosed as a case of stroke but further evaluation showed CNS citrobacter koseri infection mimicking the symptoms of stroke.

2. Case Report

A 70 year male, background of hypertension, IHD, DM on blood thinner medications presented in Department of Neu- rosurgery with severe headache for 2 days. Routine blood investigations were normal. Neurological examination re- vealed mild right sided pronator drift. CT Brain showed bi- lateral acute subdural hematomas (R>L). Patient was started on iv antiepileptics, analgesics and anti-odema drugs. Blood thinner medication was stopped and opinion from cardiolo- gist was taken for the same. 2D

echo done showed 55% EF. After 4 days of conservative management repeat CT Brain was done which showed decrease in SDH. Hower on day 6 patient had sudden onset of left sided weakness and slurring of speech. Code purple was activated and MRI Brain was urgently done considering the possibility of stroke. However MRI Brain done showed no signs of stroke and increase in SDH with 10 mm midline shift.

Patient underwent right FTP mini craniotomy and clot evacuation. On post operative day 2 patient had a seizure and hyponatremia with serum Na 127 meq/L which was managed conservatively. On post operative day 6 patient developed severe sepsis and was electively intubated due to breathing difficulty. Patient developed transient hemiparesis lasting for 24 hours. CT Brain done showed multiple hypodensity areas in frontal, temporal and parietal region. MRI Brain revealed acute cortical - subcortical infarcts in frontal and parietal lobes bilaterally and right temporal lobe with small areas in both occipital lobes and left cerebellar hemisphere

MR Angiogram showed normal circle of Willis vessels. No signs of arterial occlusion. MR Venogarm showed no dural venous sinus thrombosis. Echocardiography (LVEF 50%). LP was done. CSF showed raised count 1000 leuko- cytes (85% polymorphs), 1 mg/dl glucose and 400 mg/dl proteins.. CSF culture grew citrobacter koseri. Systemic cul- tures were sent. Patient was started on iv meropenem. On postoperative day 10 patient had a asystole for which CPR was given. Patient was revived. Patient was continued on antibiotics. Patient underwent tracheostomy and was slowly weaned off from the ventillatory support and discharged.

3. Discussion

Citrobacter koseri, is a gram negative non-spore forming, rod-shaped bacterium. It's a facultative anaerobe capable of aerobic respiration. [1] The members of this family are commensal organism and are part of the normal flora. It's commonly found in the digestive tracts of humans and ani- mals. [2]. Infections caused by C. koseri can lead to various symptoms, including fever, chills, diarrhea, and abdominal pain. In severe cases, the bacterium can cause sepsis, menin- gitis, or brain abscesses. [3,4] Arterial and venous infarctions are possible because of the bacterial infiltration along the main vessel; exudates within the ventricles and ventriculitis may obstruct the ventricular foramina and result in mul- ticystic hydrocephalus with consequent long-lasting shunting difficulties and necrotizing meningoencephalitis with pneu- mocephalus has been reported. [5] Early and massive tissue necrosis is a specific feature of *C. koseri* brain infection. The early stage of the disease predominates in the white matter, causing cerebritis; the later stage is marked with necrotic cavities in multiple locations. In samples collected from cer- ebrospinal fluid, *C. koseri* grows well on any ordinary medi- um; they produce unpigmented, colorless mucoid colonies. If incubated for 24 hours in other media such as indole, citrate, and adonitol, *C. koseri* will be positive, hydrogen sulfide negative in Kligers' iron agar, and negative results in lactose, salicin, and sucrose broth as well. [6] A broad spectrum cephalosporin and meropenem are often used because of the good penetration into the central nervous system. [7, 8] The prognosis of the *C. koseri* infection is 20 to 30% of neonates die, and 75% of survivors have significant

neurologic dam- age such as complex hydrocephalus, neurologic deficits, mental delay, and epilepsy [5]. Severe sepsis induces brain hypoperfusion. [9] The acute dysregulated inflammatory response that is meant to eradicate the infectious agent in patients with sepsis may cause hypercoagulability and vasculopathy of cerebral blood vessels leading to cerebral ische- mia. Acute vascular endothelial dysfunction is a central event in the pathogenesis of sepsis, contributing to vascular permeability and impaired autoregulation of cerebral blood flow [10].

4. Conclusion

Early diagnosis and treatment is crucial in CNS infections. Diagnosis of CNS infections becomes extremely challenging when the patient with severe systemic infection present with stroke like symptoms. Stroke mimics are common.. Possibil- ity of active infection should be considered while dealing with stroke like presentation. Commensal organisms causing life threatening infection warn us about the new emerging life threatening pathogen in neurosurgery.

Abbreviations

CNS Central Nervous System

CPR Cardiopulmonary Resuscitation CT Computed Tomography

DM Diabetes Mellitus

IHD Ischemic Heart Disease

MRI Magnetic Resonance Imaging SDH Sub Dural Hemorrhage

Declarations

Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Ong CL, Beatson SA, Totsika M, Forestier C, McEwan AG, Schembri MA (2010). "Molecular analysis of type 3 fimbrial genes from Escherichia coli, Klebsiella and Citrobacter spe- cies". BMC Microbiol. 10: 183. https://doi.org/10.1186/1471- 2180-10-183
2. Species Citrobacter koseri". LPSN. Retrieved 21 April 2023.
3. Pennington, Kelly; Van Zyl, Martin; Escalante, Patricio (6 October 2016). "Citrobacter koseri Pneumonia as Initial Presentation of Underlying Pulmonary Adenocarcinoma". Clinical Medicine Insights: Case Reports. 9: 87–89. https://doi.org/10.4137/CCRep.S40616

4. Greenwood, David; Slack, Richard C. B.; Peutherer, John F.; Barer, Michael R. (2007). Medical Microbiology: A Guide to Microbial Infections: Pathogenesis, Immunity, Laboratory Di- agnosis and Control (17th ed.). Elsevier. pp. 1264–6. ISBN 978-0-7020-4009-2.
5. Babyn, Paul S. (2011). "Section I: Neuroradiology: Case 3". Teaching Atlas of Pediatric Imaging. Thieme. pp. 44–7. ISBN 978-1-60406-494-0.
6. Doran TI (1999). "The role of Citrobacter in clinical disease of children: review". Clin. Infect. Dis. 28 (2): 384–94. https://doi.org/10.1086/515106
7. Jump up to: [ab] Feferbaum R, Diniz EM, Valente M, Giolo CR, Vieira RA, Galvani AL, Ceccon ME, Araujo MC, Krebs VL, Vaz FA (2000). "Brain abscess by Citrobacter diversus in infancy: case report". Arq Neuropsiquiatr. 58 (3A): 736–40. https://doi.org/10.1590/s0004-282x2000000400023
8. McPherson C, Gal P, Ransom JL (2008). "Treatment of Citrobacter koseri infection with ciprofloxacin and cefotaxime in a preterm infant". Ann Pharmacother. 42 (7): 1134–8. https://doi.org/10.1345/aph.1L008
9. Pfister D, Siegemund M, Dell-Kuster S, Smielewski P, Rüegg S, Strebel SP, Marsch SC, Pargger H, Steiner LA. Cerebral perfusion in sepsis-associated delirium. Crit Care. 2008; 12(3): R63.
10. Taccone FS, Castanares-Zapatero D, Peres-Bota D, Vincent JL, Berre J, Melot C. Cerebral autoregulation is influenced by carbon dioxide levels in patients with septic shock. Neurocrit Care. 2010; 12(1): 35–42.

Figures



Figure 1

CT Brain (P) showing bilateral acute subdural hemato- mas.

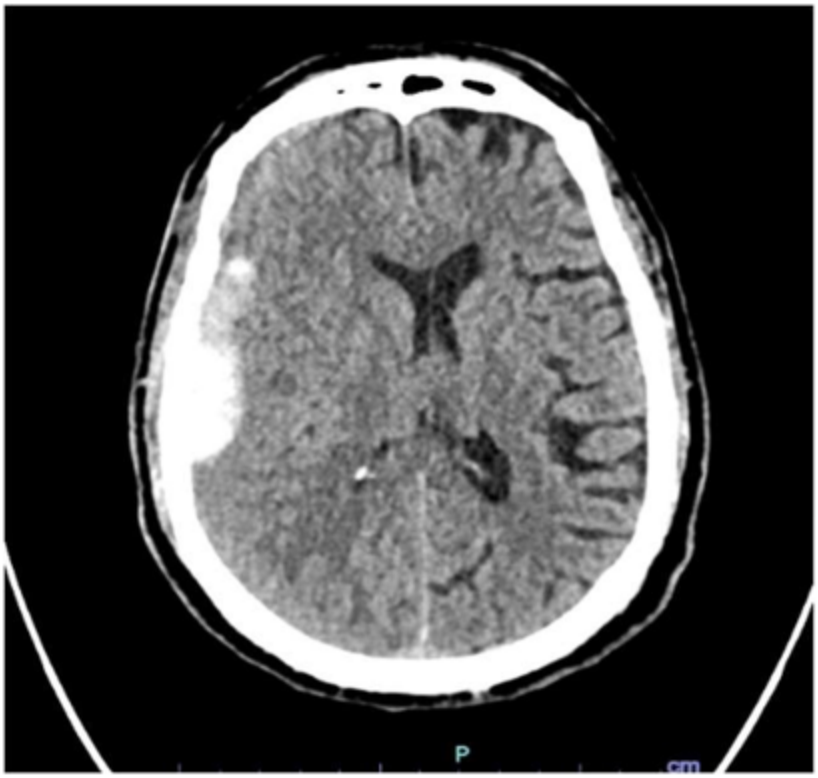


Figure 2

As compared to figure 1 bilateral subdural hematomas shows reduced in density without progression in size.

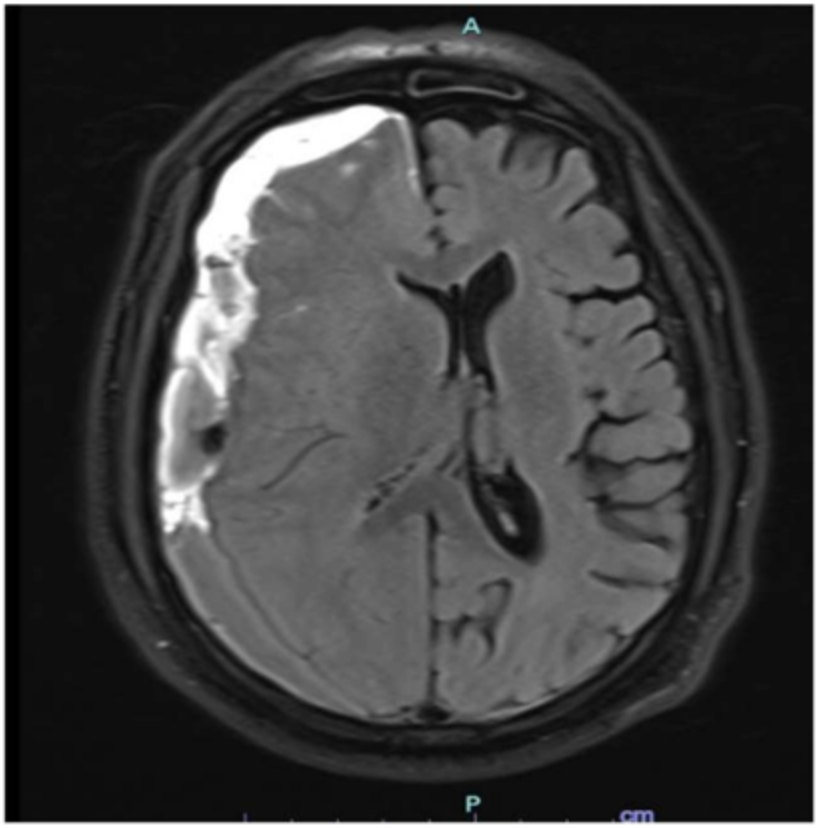


Figure 3

MRI Brain showed increase in size of the right cerebral convexity subdural hematoma with interval increase in the midline shift (now 10 mm to the left).



Figure 4

CT Brain showed areas of cortical and subcortical hypo- density in the right parietal region and left frontoparietal region causing blurring of the gray-white matter interface.

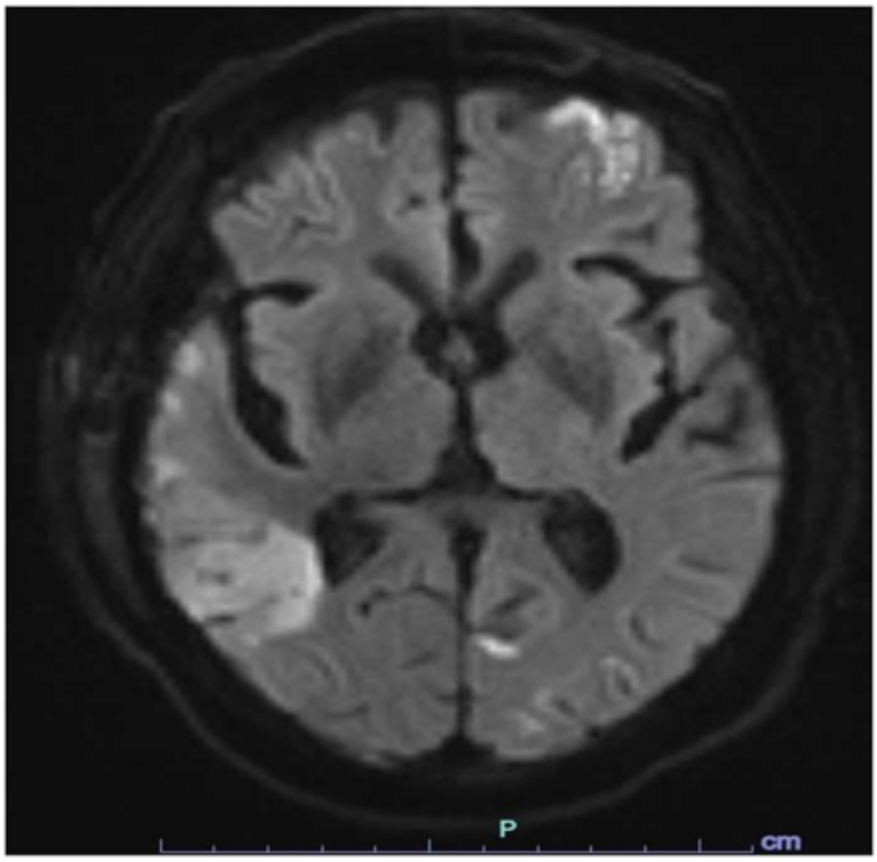


Figure 5

Multiple patchy acute cortical - subcortical infarcts with FLAIR hyperintensity in frontal and parietal lobes bilaterally and right temporal lobe with small areas in both occipital lobes and left cerebellar hemisphere.